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DELAWARE STATE MEDICAL JOURNAL

Official Organ of the Medical Society of Delaware

INCORPORATED 1789

VOLUME VI
NUMBER 9

SEPTEMBER, 1934

Per Year $2.00
Per Copy 20c

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Entered as second-class matter June 28, 1929, at the Post Office at Wilmington, Delaware, under the Act of March 3, 1879. Business and Editorial offices, 1022 Du Pont Bldg., Wilmington, Delaware. Issued monthly.

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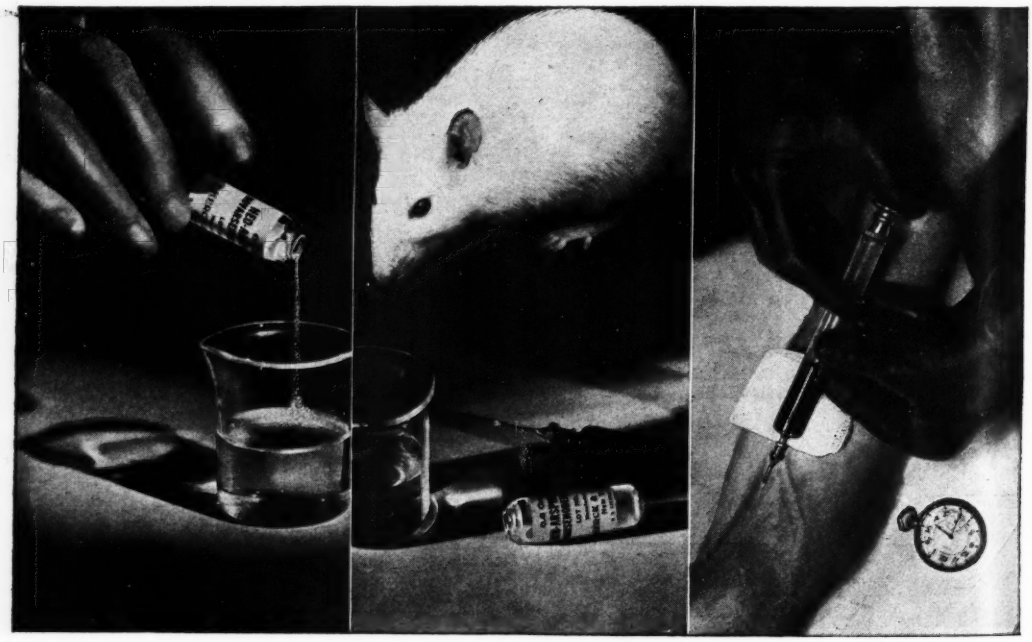

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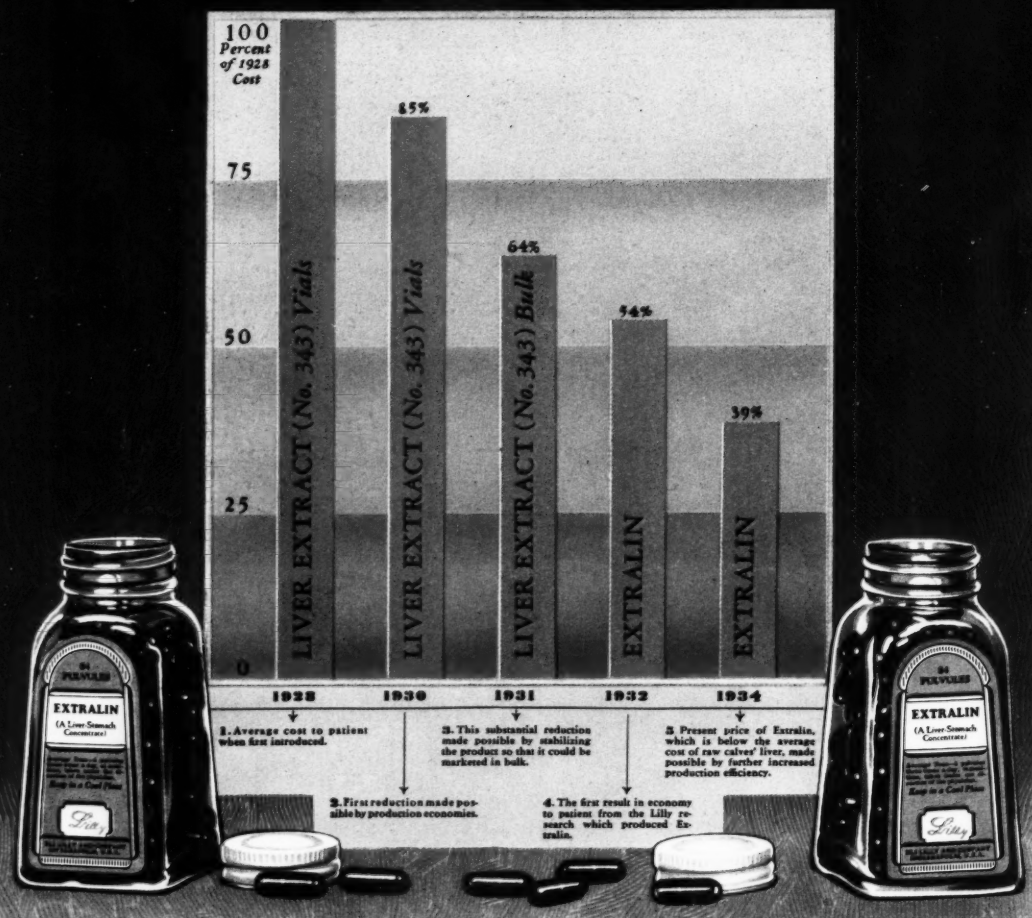

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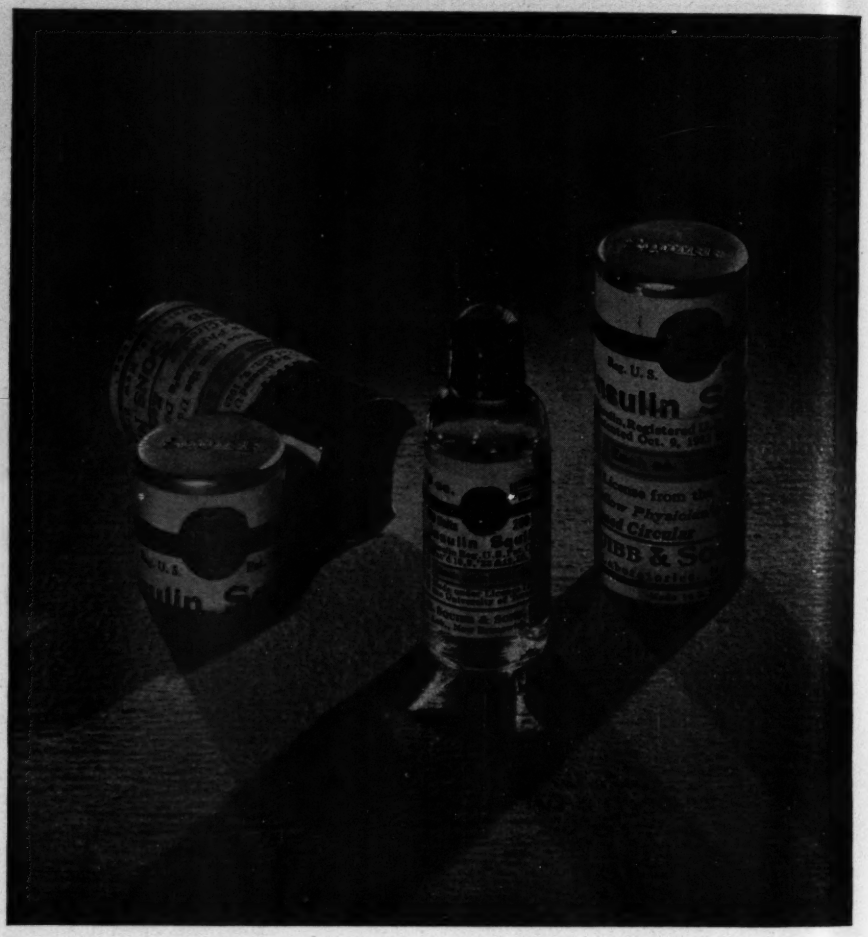

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DELAWARE STATE MEDICAL JOURNAL

Owned and Published by the Medical Society of Delaware

Issued Monthly Under the Supervision of the Publication Committee

Volume VI
Number 9

SEPTEMBER, 1934

Per Year $2.00
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IMPAIRMENT OF HEPATIC CARBOHYDRATE FUNCTION ESTIMATED BY LEVULOSE AND GALACTOSE*

By ROBERT W. TOMLINSON, M. D.**

INTRODUCTION

Prior to inaugurating the experimental work incident to the evaluation of the respective merits of levulose and galactose as integers of hepatic dysfunction, it was necessary to review the literature in English, recounting their employment in such capacity. The various antecedent writers have been quoted freely and verbatim, and to obviate the charge of plagiarism, credit has been accorded to the several experimenters and their work by parenthesised numbers, referring to the appended list of the literature reviewed.

If the effort to present with minutia of detail the conclusions, previously expressed by various investigators, and to record the results accruing from personal research has caused this paper to be unusually long, such has been attributable to a desire to pursue such experiments with an open mind, devoid of any semblance of preconceived bias.

The writer expresses his thanks to Drs. Harry L. Bockus, Joseph Bank, Harry Tumen, and H. N. Metzger, of the staff of the Graduate Hospital for their kindly and esteemed advice; to Messrs. Keller and Burgoon, and the young lady technicians of the Bio-Chemical Laboratories and to the resident physicians and the nursing staff of the Graduate Hospital for their co-operative helpfulness, without whose assistance it would have been more difficult to have attained the results accrued.

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**Thesis submitted to the Faculty of Internal Medicine of the Graduate School of Medicine of the University of Pennsylvania in partial fulfillment of the requirements for the degree of Master of Medical Science (M. Sc. (Med.)) for graduate work in Gastroenterology.

THE FATE OF INGESTED CARBOHYDRATE (Peters and Vanslyke) (22).

I. Behavior of different carbohydrates in the alimentary canal

Of the carbohydrates of the usual foods, some are celluloses and pentosans entirely incapable of absorption, others are polysaccharides capable of hydrolysis by the digestive processes into monosaccharides that can be absorbed, while others are already monosaccharides capable of absorption without hydrolysis.

The assimilable polysaccharides, starch, glycogen of meat, lactose of milk, sucrose and maltose, are all rapidly hydrolyzed by the various digestive enzymes to their constituent monosaccharides, which are then absorbed into the portal circulation. Glucose constitutes a large portion of the monosaccharides; all that formed from starch, glycogen and maltose, and half that from sucrose and lactose. The remainders of the latter two disaccharides consist of fructose and galactose respectively.

It was at one time believed that all sugars were converted into glucose in the course of their passage from the intestine to the portal circulation. It is now known that monosaccharides of all kinds are absorbed as such into the blood stream and are only converted to glucose after they have reached the liver.

The amount of sugar or the concentration in which it was given affected only the duration, not the rate of absorption. Monosaccharides are absorbed into the blood stream without transformation. After their ingestion, therefore, unless they are immediately removed from the blood stream, they will be

found in the circulating blood. As reducing agents, all will react to the tests usually employed for the estimation of glucose in the blood. The only differences in reactions to alimentary and parenteral administration of such sugars depend on, (1) the limited rate at which they can be absorbed from the intestines, and (2) the fact that sugar absorbed from the gut must traverse the liver before it gains access to the systemic circulation.

The appearance of melituria (sugar in the urine) following the ingestion or injection of monosaccharides cannot be interpreted as glycosuria (glucose in the urine) unless some method more specific than the ordinary reduction techniques is employed for its identification, unless it be assumed that the kidney is impervious to sugars, other than glucose, which it is not.

II. Removal of monosaccharides from the blood stream by simple absorption and diffusion

The injection or the absorption from the alimentary tract into the blood stream of any monosaccharide is followed for a variable interval by an increase in the concentration of sugar in the circulation. The quantity of extra sugar, which thus appears in the blood stream, must depend on the rate of administration of the sugar and the speed with which it is removed from the blood. The nature of the extra sugar is, at least at first, identical with that of the monosaccharide given and, unless the latter is glucose, qualitatively distinguished from the normal blood sugar.

The height to which the blood sugar rises must depend on the nature as well as the quantity of sugar ingested or injected. Of the three common monosaccharides, galactose causes the greatest blood sugar rise, fructose much less than galactose. This is at least partly to be ascribed to the fact that galactose is the most and fructose the least rapidly absorbed from the alimentary tract.

III. The conversion of carbohydrate to glycogen

In the normal individual, at least, retention of sugars in the tissues as monosaccharide is a minor and temporary process. Most of the sugar removed from the blood is very rapidly converted into glycogen in the liver or tissues or oxidized in the latter.

Of the various sugars or sugar-forming substances, Mann found that only glucose, maltose and glycogen (the last two polymers of glucose) could be utilized freely by the liverless animal. All other monosaccharides and compounds must, apparently, undergo transformation by the liver before they can be oxidized or converted into glycogen in the tissues. This preparatory process consists of conversion to glucose, probably through the intermediary stage of glycogen. Certainly the usual path of levulose metabolism leads through liver glycogen formation. The evidence indicates that levulose requires transformation to glycogen before it can be utilized and that this transformation is most readily effected in the liver, although it may occur to some extent in the muscles.

The speed with which different sugars can be utilized after they enter the blood stream depends on the amount of preliminary transformation which they must undergo. Glucose is the most rapidly removed from the blood stream, because it can be taken up and used by both tissues and liver. Of the other two common sugars, levulose can apparently be converted to glycogen more rapidly by the liver than galactose.

Although only glucose and its polymers can contribute directly to the glycogen stores of the tissues or to the glucose content of the blood, all glucose forming substances may, after transformation by the liver to glycogen and subsequent hydrolysis to glucose, produce hyperglycemia. There exists between liver glycogen, blood sugar and muscle glycogen, a continuous interchange governed in accordance with the resources and needs of the body. If, therefore, of some sugar other than glucose, more is taken than can be immediately retained in the liver as glycogen, the excess is converted to glucose, poured into the circulation and carried in the blood to the tissues, where it is removed for combustion or storage. The mere presence of levulose in the blood stream does not alleviate hypoglycemic shock. Nevertheless, insulin hypoglycemia may be prevented or abolished by the administration of levulose, because this increases

the supply of liver glycogen, which may be called upon to provide blood glucose.

IV. Glycogen

To Claude Bernard physiology owes the discovery of glycogen or animal starch, and its function as a form of stored energy.

V. Glycogen as a storage carbohydrate

Glycogen is non-diffusible through membranes and, like other colloids, has a negligible osmotic pressure. It, therefore, constitutes a form of carbohydrate, which can be localized in the cells in large amounts without causing them to burst by osmotic pressure. Glucose is the form of carbohydrate used for transportation, and perhaps, for immediate combustion. All of the free glucose in the body, however, might be burned by fifteen minutes of vigorous exercise, were it not renewable from the glycogen stores.

Studies of carbohydrate metabolism in man are, of necessity, based either on determinations of the carbohydrate combustion, as shown by the gas exchange, or upon glucose analyses. Clinical analyses of the quantitatively important glycogen are prevented by its inaccessible location in the tissues. Nevertheless, the intimate relationship between tissue glycogen and glucose have been sufficiently clarified by animal experiment to assist in explaining some of the multifarious variations of blood sugar. Without a knowledge of these relationships it would, in fact, be impossible to understand the behavior of the blood sugar.

VI. Distribution of Glycogen

Glycogen is the main carbohydrate of the tissues, as glucose is of the blood. Under ordinary conditions, the body probably contains ten times as much glycogen as glucose. Glycogen is found in all cellular tissues, but is most abundant in the liver, where it varies extraordinarily with the state of nutrition of the body. The liver glycogen appears to serve as a readily available reserve food for the entire body; a full supply can be piled up in a few hours in time of plenty, and in time of need can be used up with equal speed. The muscle glycogen fluctuates with the state of nutrition, but not so hugely as the liver glycogen.

SOURCES OF GLYCOGEN FORMATION

Glycogen is apparently formed in situ in each tissue. The material for its construction is absorbed by the tissue from the blood. As has been already pointed out, glycogen may be formed in the liver from glucose, other sugars, or even other substances, like protein, which are capable of conversion to carbohydrate. Other tissues can form glycogen only from glucose, which may be derived from the food, from hydrolyzed liver glycogen, or from lactic acid. Both the rate and mechanism of glycogen formation vary with the nature of the material from which it is formed.

VII. Rate of Glycogen Formation

The different sugars vary in their power to form glycogen when they are orally administered. Thus Murschhauser found that, if the increase in liver glycogen caused in eight hours by feeding 50 gm. of glucose to dogs was rated as 100, the other sugars ranked relatively as follows: levulose, 65; sucrose, 58; maltose, 16; galactose, 13; lactose, 6. This is a variance with Cori, who found far more glycogen in the livers of rats four hours after feeding fructose, than he did after glucose. On the other hand, the total amount of glycogen formed in the body was the same after either sugar. The same glycogen is formed from all these sugars. The different rates at which sugars, after ingestion, form glycogen are doubtless in part due to variations in the rates of absorption, in part to variations in rate and mode of utilization after absorption. If sugar is absorbed slowly, combustion can keep pace more nearly with absorption, a large fraction of the absorbed sugar can be burned "pari passu," and a smaller fraction remains to be stored as glycogen. However, the variation in mode of utilization after absorption appears to be an equally important factor in determining glycogen formation. Cori found that galactose was absorbed more rapidly than glucose, yet galactose feeding causes much slower glycogen formation; a larger portion of galactose must be burned or turned to fat. Fructose is more slowly absorbed than glucose, but unlike the latter,

cannot be utilized by the tissues until it has first been converted. This may explain why, in Cori's experiments, the proportion of the total body glycogen found in the liver was greater after fructose than after galactose.

VIII. Possible role of glycogen as obligatory intermediate step in glucose combustion

It is compatible with the facts known that even the fraction of absorbed glucose which is burned at once passes through glycogen as an intermediate step preceding combustion. If glucose combustion could be observed in an animal with glycogen free muscles, we should have a positive answer to the question whether glycogen formation is a necessary intermediate step.

IX. Summary of facts concerning metabolic role of glycogen

Glycogen is the carbohydrate into which absorbed sugar is immediately transformed in the tissues for storage as a reserve food. The liver is distinguished by its ability to store relatively great amounts of glycogen, which can be later broken down to glucose to maintain sugar content of the blood and to supply other tissues with energy. It is also the only organ that can form glycogen from other material than glucose or polymers of glucose. The other tissues can form glycogen from glucose and store it, but can return it to the blood only as lactic acid, which cannot be reconverted to glucose in the absence of the liver. Injuries which prevent the combustion of carbohydrate tend to deplete liver glycogen. Glycogen formation may be an essential step in carbohydrate combustion.

X. The normal sugar concentration in Venous and Capillary Blood during the post-absorptive period

The concentration of sugar in either venous or capillary blood of healthy individuals, studied before breakfast after a fast of twelve to sixteen hours, usually falls within the limits, 70 to 120 mg. per 100 cc.

XI. Diseases of the liver and gall bladder

In diseases of the gall bladder and in various pathologic conditions of the liver, excessive alimentary hyperglycemia occurs with considerable frequency.

Leclity and Woods have presented some cases in which operative relief of the gall bladder disease has been followed by restoration of sugar tolerance. It has been demonstrated that moderate liver injury raises blood sugar, indicating a loss of glycogenic function and reduction of the glycogen stores of the liver.

Because glucose tolerance tests have given such uncertain information concerning liver function in disease, other sugars have been substituted, especially levulose. It is claimed by many observers that the tolerance for levulose is more frequently diminished by liver disease than is that for glucose. The experience of Cori that levulose, when injected into the mesenteric vein, can be utilized twice as fast as when injected into a peripheral vein, yields some support to this claim. But since the muscles also are shown by Mann to form glycogen from levulose, complete loss of the liver glycogenic function would presumably only decrease, not abolish, ability to utilize this sugar; and study of the quantitative effects of partial liver destruction is necessary before the clinical value of the levulose tolerance test can be established.

EXTRACTIONS FROM A REVIEW OF THE LITERATURE

As early as 1874, Bock and Hoffman indicated the importance of the carbohydrate function of the liver. These investigators found a diminution of the blood sugar after the liver had been removed or shunted from the circulation. It remained for Mann and his co-workers to establish the importance of this function of the liver. They found that immediately after the removal of the liver, the blood sugar decreases progressively; when it reaches a certain rather definite limit, of from 0.06 to 0.04%, a characteristic group of symptoms appears and, if allowed to continue, death soon ensues, and showed that these phenomena could be controlled by the injection of dextrose, and that the duration of control, within certain limits, depended on the amount of dextrose administered, and definitely stated that the removal of the liver

completely severs a part of the vital normal process of carbohydrate metabolism. (4).

It is readily seen from a brief review of the literature that investigations of the use of sugars in testing liver function were productive of widely variant results and equally divergent interpretations. However, the fact seems to have been, that "nature," in providing for so vital a faculty, has characteristically created an organ with adequate reserve. Then, too, little consideration was given the remarkable power of regeneration of the liver cells, as a result of which, in chronic damage of these cells, one may see cellular regeneration and degeneration proceeding simultaneously. It is because of these two properties, adequate reserve and a strong tendency to regeneration, that in cases of slow or local disease of liver cells, there is sufficient actively functioning liver tissue, to maintain the carbohydrate mechanism. From these considerations, it becomes obvious, that one should expect changes in the carbohydrate function of the liver, only when all or nearly all of the liver cells are damaged more or less acutely, or when chronic damage of these cells has progressed extensively and regeneration has failed. This contention is strengthened by Roubitschek, who found marked galactosuria, but in those cases in which, despite additional poisoning by phosphorous, there was a recession of the galactosuria, post-mortem examination revealed regeneration of the liver cells. These facts, then, of adequate hepatic reserve and ready hepatic regeneration, aid in the clarification of the variable results that have attended the efforts to utilize the carbohydrate function as a routine test of liver function. (4).

Claude Bernard discovered the glycogenic function of the liver in 1857. In 1899 Sachs demonstrated that 90% of cases of liver disease showed levulosuria, whereas 90% of normals did not. Positive cases showed levulose in the urine within one hour after administration of the sugar. Strauss in 1901 first suggested the utilization of this function as a test of hepatic disease. Since the original paper much has been written concerning this test, but the majority of observers agree, that in its original form, the test is unreliable. (1-2). Shirokaeur was the first to substitute the estimation of the blood sugar in place of urinary sugar. This observer noted that there was little or no rise in the blood sugar content in a normal person, one hour after the administration of 100 gm. of levulose. If, however, the liver was damaged, the rise was marked—even up to 190 mg. per 100 cc, or if the rise above the first value was over 30 mg., a degree of hepatic inefficiency was indicated. (1-2).

A study has been made of the action of hydrazine and certain of its derivatives in producing liver injury, and the levulose tolerance test was employed in determining the degree of liver involvement, and this method has been found to be very useful, as shown by the corroborative evidence adduced on gross and microscopic post-mortem examination of the tissues of the experimental animals. (3).

"Holweg" (2) found that obstruction of the common duct by a gall stone may give rise to a positive levulose tolerance test; whereas, the obstruction of a tumor does not, even if the obstruction in the latter case becomes quite complete. Evidently the blocking of the duct which comes on gradually or irregularly, does not overpower the liver cells; so, a common duct stone, may or may not give rise to an associated hepatitis and consequent alteration of glycogenic function, and it is not primarily in obstructive jaundice that we anticipate a positive levulose test. (2).

In 1906 Baeur suggested that galactosuria, following the ingestion of galactose, might be used as a liver function test. (2).

The unreliability of the urine test in the case of levulose was shown by Lansberg in 1903, Churchman in 1912, and Woerner and Reiss in 1914. Churchman found levulosuria absent in a number of cases of liver disease, and present in a number of normals. (2).

Comparative studies of blood sugar curves on normal individuals after the ingestion of glucose, lactose and levulose, showed that the levulose produced no rise at all. (2).

Bodansky, in 1923, found that levulose produced little hyperglycemia and gave an accurate index of liver dysfunction, performing the test before chloroform poisoning, at the

height of the poisoning, and during the recovery of those animals less severely poisoned. The tolerance after chloroform poisoning returned to normal within six to fourteen days; in those animals which died, fatty degeneration and necrosis of the liver cells was demonstrated histologically. One animal subjected to chronic alcohol poisoning, showed by the liver test, moderate hepatic insufficiency. (2).

Greene, Snell, Walters, McVicar and Rowntree, in a series of papers in 1925, criticized the levulose tolerance test, because it is inconstantly present in obstructive jaundice and malignancy of the liver, and because of the difficulty in ruling out associated pancreatic disease. Experimentally, they found the test positive in a high percentage of dogs after ligation of the common duct, but obstruction of such cannot interfere with the glycogenic function of the liver, but the retention of the bile may result in injury to the liver cells. Clinically, biliary obstruction is seldom strictly comparable with experimental ligation. It is probably in only a small percentage of clinical cases, that the liver cells receive the sudden shock which comes from sudden and total ligation. (2).

There has been a great deal of investigation recently regarding hepatic function and the various tests to determine it, the importance of which is quite obvious to the Clinician and Surgeon. They aid materially in the diagnosis of hepatic disease, and also in determining the prognosis previous to Surgical procedures. It is essential to know the changes which have taken place in the liver before surgery of the gall bladder and its duct is attempted. The liver plays an important part in the general nutrition of the body. Its functions are manifold, but for the most part they depend on the properties of the liver cells, which constitute the anatomical and physiological unit of the organ. (5).

The blood sugar level depends on the relative rate at which sugar enters and leaves the blood stream. Maintenance of a constant blood sugar concentration, requires an in and out flow of sugar, of equal magnitude. When this equilibrium is disturbed the blood sugar may either rise or fall, depending on the preponderance of the processes which contribute to or cause a withdrawal of sugar from the blood. It is now generally accepted, that a rise in blood sugar, elicits a secretion of "insulin," and that a fall in blood sugar, below a certain level, is followed by a discharge of epinephrine. Two processes contribute sugar to the blood; namely, absorption of carbohydrate from the alimentary canal, and sugar formation in the liver (from glycogen and other sources). The disappearance of the blood sugar depends on its oxidation in the tissues, and upon its conversion into glycogen and fat. (7).

GLYCOGENOLYSIS IN THE LIVER

Under post absorptive conditions the organism depends on the liver for the supply of blood sugar, as is shown by the fact that removal of this organ causes hypoglycemia (Mann and McGath). Though the muscles generally contain a store of glycogen, which is at least equal to that in the liver, they are unable to contribute glucose to the blood. Since the blood sugar remains constant under normal post absorptive conditions, in spite of fluctuations in the rate of its utilization in the tissues, sugar production in the liver must be capable of very fine and rapid adjustment. The liver is able to convert a large variety of substances into glucose; in most cases this, probably, does not occur directly, but involves intermediary glycogen formation. The final step in the process of sugar formation in the liver consists, therefore, in the enzymatic hydrolysis of glycogen. Among the various factors influencing the rate of sugar formation in the liver, Insulin and Epinephrine are of chief interest. (7).

THE INHIBITING INFLUENCE OF INSULIN

Hedon and Geraud showed that increased glycogenolysis sets in as soon as the supply of insulin is stopped. Determinations of the sugar concentration in, the in and outgoing blood of the liver, before and after insulin injections, showed that less sugar is liberated by the liver during hypoglycemia, than at a normal sugar level, and the experimental evidence so far presented, is consistent with the view, that insulin acts as an inhibitor of the glycogenic process in the liver. In well-nourished animals with high liver glycogen,

if the hypoglycemia persists for some time, insulin causes a fall in liver glycogen. The decrease in liver glycogen after insulin injection, is generally associated with hypoglycemia, and there is reason for believing, that the latter is a powerful stimulant to glycogenolysis, and the fall in glycogen would, therefore, not be caused by insulin, but would be of the nature of a secondary phenomenon.

When insulin is lacking, the enzymatic hydrolysis of liver glycogen remains unchecked, leading to the disappearance of liver glycogen, and illustrating that a continuous supply of insulin, by the pancreas, is therefore essential for the preservation of hepatic glycogen, whereas one of the functions of insulin in the body is an inhibition of sugar formation in the liver. Epinephrine has the opposite effect, the injection causing a decrease in liver glycogen and an increase in blood sugar. The decrease in liver glycogen in the beginning of "epinephrine" action indicates that glycogenolysis is proceeding more rapidly than new formation of liver glycogen (from blood lactic acid), but, in later periods of epinephrine action, the reverse takes place, the rate of new formation exceeding the rate of mobilization, the end result being an increase in liver glycogen above the original level.

The presence of both glycogen and glycogenase in the same cell, makes it necessary to assume that a large part of the enzyme is unable to act on the sub-strate, otherwise the glycogen would quickly disappear from the liver. That the glycogenase can be considered more active than under "in vivo" conditions is shown by the rapidity of glycogenolysis. The enzyme may be relatively inactive in the intact liver because it requires an activator or because it is blocked by an inhibitory substance, or the enzyme may be absorbed on some structures in the cell and thus be removed from the sphere of action. The largest part of the glycogenolytic enzyme is absorbed, and only a small fraction is free, and, therefore, in an active state in the liver of a normal animal, whilst the opposite is the case in the liver of a diabetic animal. Insulin, either directly or indirectly, would cause a reabsorption of the enzyme and thus diminish sugar production in the liver; while epinephrine would increase sugar formation, by liberating part of the bound enzyme. (7).

GLYCOGEN FORMATION IN THE LIVER

When food containing carbohydrate is taken, glycogen is deposited in the liver, but even with a diet practically free of carbohydrate the glycogen stores of the liver are not depleted. This is due to the fact that a variety of non-carbohydrate substances can serve the liver as material for glycogen formation. (7).

The feeding of fructose gives substantially the same results as those obtained with glucose, if it is true that fructose is converted into glucose in the liver, and normally never gets beyond that organ, this assumption being based on the fact, that neither normal nor diabetic urines contain fructose when large quantities of such or of cane sugar are taken, and the fructose test for a much-reduced efficiency on the part of the liver rests on such assumption. Since fructose has nowhere near the same effect as has glucose in raising the level of the blood sugar, one is forced to assume that the transformation of fructose into glycogen within the liver is accomplished much more easily and, therefore, more completely. (8).

In 1921 McLean and de Wesselow studied blood sugar curves of normal adults and those of diabetics after the injection of various sugars, such as glucose, levulose, saccharose and galactose, etc. All these sugars, except levulose, provoked a rise in blood sugar. As little as twenty-five gm. of these sugars provoked a maximal rise. Larger doses were incapable of increasing the level, merely tending to prolong the time during which this level was maintained. Levulose on the contrary had no effect on the blood sugar in persons with normal livers.

Spence and Brett, later found the ingestion of fifty gm. of levulose will show no rise in blood sugar. More than fifty gm. may cause an alimentary levulosuria, because even a normal liver has a limit of storage, and all levulose above this limit will be spilt over into the blood and excreted by the kidneys. The rationale of this test, therefore, depends upon the fact that the normal liver is able to

convert and store fifty gm. of levulose given at one time. If the liver is in any way damaged, the entire amount would not be taken up by its cells, and some levulose would appear in the blood. (9).

When levulose is given by the mouth it is found a considerable dose can be tolerated without giving rise to a recognizable increase in the blood sugar. All of the levulose which is absorbed from the intestine, must first pass through the portal system to the liver, before it can gain entrance to the systemic circulation. The natural interpretation of these facts is, that the liver has the power of building up and storing levulose. In fact, as Sir Humphrey Rolleston has noted, levulose is the only carbohydrate, of which the metabolism cannot be carried on elsewhere in the body, than in the liver. It may be stated in brief, therefore, that normally there is little or no rise in the blood sugar curve following the ingestion of a given amount of levulose. The sequence of events is far different, however, in the case of gross liver disease whether due to degeneration, atrophy, inflammation or new growth. In these circumstances, the liver cells are either partly destroyed or rendered individually less capable of function. They are, therefore, unable to deal efficiently with the levulose after it is absorbed from the intestine. Consequently the sugar evades polymerization and escapes from the portal into the systemic circulation, there giving rise to a marked increase in the blood sugar content, the extent and duration of which may be regarded as indicative of the degree of liver damage.

It is clear that three factors, apart from the activity of the liver, will tend to influence the height obtained by this blood sugar curve.

I. In the first place the rate of absorption from the gut must be considered. In cases where chronic inflammation or atrophy of the mucous surfaces is present, the rate of absorption will be correspondingly slow, and the concentration of levulose in the blood, at any given time, will be relatively lower than in cases with healthy mucous membrane.

II. The pancreas has a very intimate connection with certain stages in the metabolism of sugars in the animal body; clearly, then an anomalous result might be obtained by exhibiting levulose in a case of pancreatic deficiency. This possibility must be excluded, if need be, by separately determining the glucose tolerance, and by finding in the urine a diastase content of over fifty units (Davies). A reaction to levulose can only be taken as abnormal, if there is no evidence pointing to pancreatic disorder.

III. The rate of elimination of the sugar from the blood must have a definite reaction upon the height of the levulose curve. Elimination is mainly effected by the kidneys, and in some cases is much more rapid than in others. In passing, it may be noted that the threshold for levulose is considerably lower than that for glucose. Levulose can be detected in the urine after quite small degrees of rise above the fasting level of blood sugar.

However scant our knowledge of the normal fructose metabolism may be, it is certain that the greater part of the fructose which the liver gets from the intestine is stored in that organ as glycogen, and that a smaller part passes over into the blood stream from which it disappears rapidly. We also know that a part of the fructose is at once oxidized. This oxidation process is manifested by a rapid increase in the excretion of CO_2, together with a rise of the respiratory quotient, a phenomenon repeatedly observed. When the function of the liver is intact, a partial decrease in tolerance to fructose is never met. (12).

Worner and Reiss carried out experiments on alimentary galactosuria and levulosuria, and came to the conclusion, in regard to the latter, that its value as a test for liver disease could only be accepted if more than 0.1 gm. was found in the urine, and even then, the interpretation of the result needed caution.

In 1913 Shirokaeur, after giving one hundred gm. of levulose, found the rise in the blood sugar of normal individuals to show a marked difference from that following a dose of glucose. Instead of a value of 0.15 or so being obtained, only the slightest rise in blood sugar value was noted. In the subjects of liver diseases, however, the rise was marked, values up to 0.19 being observed. At the same time he found levulose in the urine in the majority of cases, though this did not of neces-

sity, show a parallel with the rise in blood sugar values.

Isaac conducted a series of experiments regarding the position of fructose in carbohydrate metabolism, not only confirming the fact that fructose, as compared with glucose, caused no appreciable rise in the blood sugar content, but claims to have estimated the relative amounts of levulose and dextrose in the blood after the oral administration of one hundred gm. of levulose. He found in samples of blood taken from normal persons half an hour, and in other cases one hour after administration of this sugar, that dextrose formed by far the greater portion of the total blood sugar content, and that levulose soon disappeared entirely. In cases exhibiting hepatic diseases, on the contrary, not only did the blood sugar content rise to a much greater extent, but the proportion of levulose actually present in the blood sugar was far greater and persisted longer.

This test is dependent on the fact that levulose is unable to cause a rise in blood sugar such as takes place after a dose of glucose. That the liver is concerned in this must be accepted, if the test is to be relied upon, as one for hepatic function.

Glucose is known not to be assimilated and stored as quickly as levulose. Even in a healthy person, its increase in the general circulation is observed by following the blood sugar curve over a period of one and a half hours. Winter and Smith have claimed to show that the form in which it circulates is Y glucose, this being derived from a common enolic form. The production of Y glucose is essential before assimilation can take place. Since fructose is more easily converted into this enolic form than glucose, it is reasonable to suggest, that the conversion and subsequent storage of fructose takes place more rapidly than in the case of glucose. Consequently, levulose does not appear in the circulation to the same extent as glucose, immediately following its ingestion. If, however, a pathological condition of the liver exists, then the enzyme or substance causing this conversion may be less active or so delayed in its action that the fructose, instead of undergoing a rapid conversion into glycogen, as is usually the case, circulates in the blood and gives rise to the blood sugar curve observed in pathological conditions of the liver. (15).

Mann and Magath found that complete removal of the liver prevented additions of glucose to the blood from other parts of the body. Unless glucose, or some other substance easily convertible to glucose, such as glycogen, starch, maltose and so forth, was administered to the animal without a liver, the blood glucose gradually lessened and symptoms of hypoglycemia ensued. Administration of substances convertible to glucose prevented symptoms of hypoglycemia and increased the glucose content of the blood.

With fructose it was demonstrated that in the absence of the liver, its administration prevented symptoms of hypoglycemia and increased the glucose content of the blood. This appears to be direct evidence that tissues other than the liver are able to effect this transformation. From a quantitative standpoint, about the same percentage of administered levulose disappeared and was converted to glucose, whether the liver was present or not. If both liver and intestines were absent, no conversion of fructose to glucose occurred, symptoms of hypoglycemia developed, and large amounts of fructose could be recovered unchanged in the urine. If only the intestines were removed, the liver being present, fructose was converted into glucose. It would appear that fructose may be converted to glucose by the liver or by the intestines, but not by the other tissues of the body.

The observations of Hughes and Malik (1930) on a number of hospital patients in Lahore (India) showed that the levulose tolerance test was positive in most of them, though there was no clinical evidence of hepatic disease. They suggested that the diminished tolerance for levulose in these patients was the result of defective formation and storage of glycogen in the liver, probably due to previous attacks of malaria, from which they had suffered.

Our results show that the levulose tolerance test gave positive results in a greater number of cases with liver disease than in those without evidence of hepatic disorder. Moreover,

while a positive test is more in favor of hepatic dysfunction, a negative test cannot exclude liver disease, especially in chronic cases. Clinical evidence of disease of the liver is generally present by the time the levulose tolerance test gives positive results, and the test then is more confirmatory, rather than an aid in diagnosis. Tests of this type depending on the partial function of the liver, in the regulation of the blood sugar level, "do not permit of an exact quantitative estimate of the degree of impairment or liver function" (De Wesselow 1924). (20).

There is a vast difference between one-fifth of a healthy liver and an entire liver, all of which is diseased, however mild that disease may be. Compare a case of embolic nephritis with a case of acute hemorrhagic nephritis. Similarly, a toxic heptatitis generally involves all of the parenchyma of the liver, and a chronic heptatitis continues to involve the entire parenchyma, whether it be made of new grown cells or old. Even if such involvement were slight, the function of the involved cells could hardly be expected to equal that of a small but normal group of cells. Mild and chronic forms of hepatitis or hepatic insufficiency do exist, and they are quantitatively measurable by the levulose test. (2).

Joliffe concluded, "that the height of the rise following the ingestion of sugar is in inverse proportion to the level of the fasting blood sugar, and that there must be a ceiling level, which is constant, whatever the fasting level, which when reached, stimulates a subsequent fall, and that the fasting level must be considered before declaring any rise in mg. to be an abnormal one." He believes that the ability of various sugars to produce hyperglycemia varies directly with the rate of absorption and inversely with the ability to form glycogen and the ease of being oxidized. Levulose is absorbed slowly, but being a good glycogen former and readily oxidized, causes only a slight rise in blood sugar, "the rise is in inverse ratio to the fasting level (the lower the fasting level, the higher the rise), and attributes the regulation of the blood sugar level to its concentration, and in line with Pollack's assertion that the blood sugar level regulates the metabolism of carbohydrates, an idea quite in line with the cause and effect of insulin secretion cited by Cori."

The active agent of this sugar storage mechanism is insulin, and it has been shown with crystalline insulin there is a latent period of ten to fifteen minutes, which elapses after injection of insulin, before the blood sugar begins to fall. Thus it is probable, that in the normal body an interval of ten or fifteen minutes elapses, after the excitatory ceiling has been reached, before the sugar storage mechanism begins to effect a significant fall in the blood sugar level, and during such interval, in a tolerance test, sugar is being constantly absorbed. Cori and Cori have shown that a single sugar is absorbed at a constant rate from the intestine regardless of its concentration and that this absorption has no relation to insulin. Since the carbohydrate storage mechanism is latent, this absorbed sugar accumulates in the blood. The more rapidly absorbed sugar may be expected therefore, to raise the blood sugar well above the ceiling level.

Sachs and Blumenthal, and more recently Mann, Magath, Bollman, Ringold and Karr, Ludhardt and Koppany, have found that levulose was absorbed almost specifically by the liver, and according to McLeod, a diabetic liver can convert levulose into glycogen, which then becomes available as glucose to the organism. Wierzuchowski and Cori have shown that action of insulin though markedly affecting the tolerance to glucose, will not, in any way, alter the tolerance to levulose (rather large quantities) administered by intravenous injection. As Cori has shown, insulin action is chiefly concerned with peripheral metabolism (increased oxidation and the formation of glycogen in muscle), whereas levulose is primarily subject to the liver, enunciated as primarily, for he has also shown that under certain conditions, the tissues can utilize levulose.

To summarize, it is plain that levulose may be metabolized outside the liver and it is equally evident that the liver rapidly and efficiently utilizes a large proportion of the levulose, which comes to it having an extraordinary affinity for levulose, and being further assisted by the fact that levulose is absorbed

at but half the rate of glucose, and any levulose which does get through the liver will then be in the systemic circulation, and the S shaped curve for levulose utilization by the liver demonstrates that there is a delay before the liver begins to store sugar, thus affording opportunity early in the absorption period for levulose to get through the liver into the systemic circulation and it is presumably this sugar which causes the rise during the levulose tolerance test. If the liver were damaged, still more would pass through and the level would be raised further to the point of giving a positive test. Any metabolism which may go on in the tissues would remove sugar from the blood, and not add to it, it having been conclusively shown (Mann, Cori), that only absorbed sugar and liver glycogen can serve as sources of sugar in the blood, never muscle glycogen. Of the three major influences known to affect the deposition of glycogen in the liver, configuration of the sugar molecule is certainly the most important and the rise of blood sugar in animals after the injection of galactose is much higher than after levulose, yet but little glycogen is deposited in the liver. The rate of absorption undoubtedly helps to determine the height of the blood sugar. In the case of levulose, all values are usually within the normal range, and the slow absorption plus the special affinity of liver tissue for levulose, must be the chief factors involved.

Technique and Methods Employed

Shirokauer and Isaac employed doses of one hundred gm. of levulose and other workers have used up to 50 gm. Spence, Brett and Tallerman varied the dosage according to the body weight, but the latter pointed out that because his results were strictly comparable with those using doses of one hundred gm., he could see no reason for not employing a constant dose of forty-five gm. and his opinion has been accepted. Scharff made a study of the effect of different doses and decided that increased doses did increase the height of the curve, but why this should be so, is difficult to see, as Cori and Cori have shown that absorption of levulose, as of any single sugar, continues at a constant rate regardless of the concentration, and increased doses merely prolong the absorption period and the most plausible explanation is a difference in behavior between animals and man, such as has been shown to exist in the case of galactose.

Study of the values quoted from Spence and Brett and Tallerman show that the first and second hour samples indicate the full extent of the curve, though missing a slightly higher rise in the first one-half hour. The one-hour sample seems the more reliable, as variations in gastric emptying and intestinal absorption may affect the height of the one-half hour level.

Various Standards of Normality

The chief conceptions in the literature, concerning the limits of normal tolerance to levulose have been presented, and liver damage has been presumed under the following conditions:

I. Rise of over ten (10) mg. per cent above the fasting level, with failure to return to the fasting level by the end of the second hour (Spence and Brett on a basis of five (5) cases).

II. Rise of over thirty (30) mg. per cent above the fasting level and to a level of over 0.135%, also persistence of a high level at the end of one and a half (1.5) to two (2) hours. (Tallerman on the basis of fifteen (15) cases).

III. Rise to a level of over 0.125% (Joliffe on the basis of eighty-one (81) cases).

Elmer and Scheps believe the highest rise in normals to be 20 mg. per cent above the fasting level. (2).

I. In normal subjects the maximum blood sugar following administration of thirty (30) to fifty (50) mg. (based on body weight) of levulose, by mouth, rarely exceeds 115 mg. per cent.

II. A rise in blood sugar to 125 mg. per cent or over, should be considered an abnormal response to the oral administration of levulose.

I. A blood sugar value of 0.12 to 0.13% after a dose of levulose is not in itself evidence of a pathological condition. If, however, the height of the curve exceeds 0.135%, and the actual rise in the value from the original level exceeds 30 mg. a degree of liver inadequacy is presumed. The prolongation of the curve, a high blood sugar level persist-

ing at the end of 1.5 to 2 hours, is strong evidence of such disorder.

II. Dosage, according to weight appears to be a questionable factor in the test. 45 gm. of levulose seems to be satisfactory as a routine dose.

III. The renal threshold for levulose is normally far lower than is the case with glucose, and can be placed at a blood sugar value of 0.12%. Since 0.1% blood sugar is normal, the increase above this, which follows the ingestion of levulose, causes the glycosuria. The active threshold for fructose is, therefore, about 0.02%. (24).

M. V. Radhakrishna Rao believes: (21)

I. That the levulose tolerance test is to be considered negative, if the maximum rise of blood sugar does not exceed 20 mg. per cent, and positive if it exceeds 30 mg. per cent.

II. If the maximum rise of blood sugar is in between these two readings (i. e.—between 20 and 30 mg. per cent), the test is considered doubtful.

According to Stockton Kimball—(Guy's Hospital—Reports, London, ——— 2.), it is apparent that there is, as Joliffe stated, a very definite difference in the rise and fall, depending on the fasting level. The group with a fasting level below 100 mg. per cent shows a tendency to a higher rise in the first hour and a less pronounced fall in the second, than is shown by the group with the fasting level of 100 mg. per cent or over.

For normals, the principles of response may thus be stated, "the lower the fasting level, the higher the consequent rise"—any rise in excess of the average during the first hour is followed by a prompt fall to fasting or below during the second. It is worthy of note that in no case in which the rise during the first hour reached or exceeded a level of 115 mg. per 100 cc of blood, was there a failure to return, during the second hour, to or below the fasting level.

A definite contrast, then, is shown between the behavior of the groups with the fasting level below 100 mg. per cent and those with the fasting level of 100 mg. per cent or over.

It is apparent that a low fasting level allows of a higher rise and slower fall, but that these effects do not always follow. One should, therefore, consider the fasting level, when deciding whether or no, tolerance is impaired. It is the sugar curve with which we are dealing, both the rise and fall must, therefore, be noted. It is more reliable to judge by the extent of this rise and fall, than to set a fixed sugar level which must be reached before a test may be called positive, and which can, therefore, never be surpassed by a normal person. There are positives which do not reach Tallerman's level of 135 mg. per cent, and apparent normals which overreach Joliffe's level of 125 mg. per cent. The following are the criteria of hepatic insufficiency finally reached on the basis of the study of normals.

I. Elevation of 30 mg. per cent or more above the fasting level within the first hour, or over 15 mg. per cent in the second hour.

II. Elevation from a fasting level of below 100 mg. per cent to within five of either of these figures, or from a fasting level of 100 mg. per cent or over, to within 10 of either of these figures, may be considered suspicious, but not diagnostic of hepatic insufficiency.

III. If, however, a suspicious level reached during the first hour, is accompanied by a suspicious level for the second, the test is to be considered positive.

Numbers II and III of the above-mentioned criteria may be stated differently, thus—"an one hour rise of 25 to 30 mg. per cent from a fasting level of 100 mg. per cent or above, if accompanied by a failure of the second hour level to drop to below 10 and 5 mg. per cent, respectively, of the fasting level, is diagnostic of hepatic insufficiency. If either the first or second hour reading is thus affected, but the other is within normal range, the test may be regarded as suspicious. (2).

The following cautions are necessary. (2).

I. If the fasting level is below 80 mg. per cent, no positive conclusions can be drawn from the curve.

II. A positive diagnosis can never be safely made upon an elevated second hour level alone, unless the first hour level, has reached at least 10 mg. per cent above the fasting level. If this caution is not observed, the

effect of a possible delay in absorption cannot be excluded.

III. If the fasting level is 120 mg. per cent or above, the greatest care must be exercised in drawing conclusion, that a Cirrhotic liver or one badly out of function will not metabolise any carbohydrate properly. Observation of cases which showed a levulose curve, all readings of abnormal range, but which yet had a return of the test to within normal limits after treatment, revealed that liver disease alone may produce a test of this type, and one is always faced with the difficulty of distinguishing the condition from diabetes. It will be seen it is not safe to make a diagnosis on the basis of a levulose test, in which all of the values remain 120 mg. per cent or above. If, however, the fasting level is below 120 mg. per cent, or the fall during the second hour is to a level below 120 mg. per cent, one may accept the test as valid.

In evaluating suspicious tests, the conception of the whole curve should be kept in mind. If the level reached during the first hour is suspiciously high, but is followed during the second hour by a sharp fall to below the fasting level, the test is presumably normal. The less sharp the second hour fall, the more should the test be considered significantly abnormal. (2).

(To Be Concluded Next Month)

CANCER COMMENT

Pathology as an Aid to Cancer Control

MARION L. H. FREEMAN*

The chief object of the American Society for the Control of Cancer has been the dissemination of knowledge regarding cancer. This has resulted in an increasing number of patients seeking medical advice regarding small suspicious lesions. With this trend in the cancer situation, the physician finds it increasingly difficult to diagnose cancer, for the earlier a suspect presents himself the less is the distinction between the malignant and the benign. The Delaware Committee has anticipated a more concerted effort in cancer control by organizing five tumor clinics, reported in THE JOURNAL last month, and by establishing a tissue diagnosis service whereby any Delaware physician in good standing may submit tissue which he suspects of being tumor to the pathologist of the Committee and receive a diagnosis.

From February 14, 1933, when the first specimen was sent in, to July 19, 1934, a total of 184 specimens have been submitted, of which 68 proved to be malignant. Physicians wishing to avail themselves of this service should place the material in 10% formalin as soon as possible and mail it to The Tissue Diagnosis Service, 14th & Washington Streets, Wilmington. It is expected that the physician will co-operate by sending the patient's name, address, age, a brief clinical record, and statement of operative findings, and where possible a contribution of one dollar or more from the patient to cover the technical and secretarial costs. It is very satisfying to note that thus far the service is nearly self-supporting.

The method of diagnosis by microscopic examination of biopsy specimen is now very extensively used, especially since there is abundant evidence to prove that most cancers may be subjected to biopsy without jeopardizing the prognosis if treatment is started within four days. The biopsy specimen need not be large—very satisfactory specimens of bladder tumors are obtained by means of a Bradsford-Lewis rongeur forceps through the cystoscope—but it is important to select the proper site. The margin of the tumor with a bit of adjacent normal appearing tissue is best. Tissue excised with the scalpel is preferable to that removed by electric resection because the unavoidable heat incident to the latter method may distort the cells most necessary to make a diagnosis. The site of biopsy may be coagulated subsequent to the excision.

The method of obtaining material by means of aspiration is especially adaptable to tumors beneath the surface. As described by Stewart in The American Journal of Pathology, 9, 801, 1933, the skin is infiltrated with novocaine and a small puncture wound made. An 18-gauge needle attached to a Record syringe is then inserted with the piston closed until it is felt to enter the neoplasm. A

(Continued on Page 218)

*Secretary-Technician of the Delaware Tumor Clinics.

The President's Page

To the Members of the Medical Society of Delaware:

Allow me at this time to thank you, one and all, for the co-operation you have given me towards the betterment of our State Medical Society in 1934.

Since being inducted into the office of President I have visited the county societies in all three counties, and have been very much pleased with the attendance, as well as with the interest shown at each meeting. The programs were very interesting and instructive and I am sure everybody has benefited by them.

We also had a midyear meeting of the State Society in Dover in April, with a large attendance and a very good program. Though the Legislature has been in session since our last yearly meeting, medical matters were not brought before them because they were too busy with serious financial problems which they were supposed to have settled. Therefore, our Society was not called upon to guard medical legislation.

Next month, October 9th to 10th, the Medical Society of Delaware will hold its annual meeting in the new State House at Dover. May I ask as a special favor of very member to please set aside those days to be present in Dover and make this meeting a grand success? An exceptionally fine program is arranged. Our local committee on arrangements has tried to make things pleasant and appetizing for all. On Tuesday evening, October 9th, Mrs. McDaniel, who is president of the Auxiliary, and we wish to extend to every member of both organizations an invitation to be our guests at an informal house-warming at our home, 229 South State street. So come to Dover Tuesday prepared to spend the night.

Hoping to see a large attendance and thanking you in advance for whatever you may do towards making our 1934 meeting a success, I am

Yours very sincerely and fraternally

JOSEPH S. McDANIEL.

EDITORIAL

DELAWARE STATE MEDICAL JOURNAL

Owned and published by the Medical Society of Delaware. Issued about the twentieth of each month under the supervision of the Publication Committee.

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All correspondence regarding editorial matters, articles, book reviews, etc., should be addressed to the Editor. All correspondence regarding advertisements, rates, etc., should be addressed to the Business Manager.

Local news of possible interest to the medical profession, notes on removals, changes in address, births, deaths and weddings will be gratefully received.

All advertisements are received subject to the approval of the Council on Pharmacy and Chemistry of the American Medical Association.

It is suggested that wherever possible members of the State Society should patronize our advertisers in preference to others as a matter of fair reciprocity.

Subscription price: $2.00 per annum in advance. Single copies, 20 cents. Foreign countries: $2.50 per annum.

VOL. VI SEPTEMBER, 1934 No. 9

ON TO DOVER

The One Hundred and Forty-fifth Annual Session of the Medical Society of Delaware will be held in the new State House, at Dover, October 8th, 9th and 10th. The House of Delegates meets on the evening of Monday, the 8th. The scientific sessions begin the morning of Tuesday, the 9th, and conclude in the afternoon of Wednesday, the 10th. An excellent program has been arranged, which will bring pleasure and profit to the profession.

For the ladies—and the men—there will be a house-warming at the home of President McDaniel on Tuesday evening. The annual meeting of the Auxiliary will be held on Wednesday, the 10th.

Every member of both the Society and the Auxiliary should make a special effort to attend the session this year. This duty especially devolves upon the Wilmington members who owe it to themselves and to their organizations to make these down-State meetings as successful as the ones held in Wilmington. Wilmington and vicinity has over one-half of the members of the Society, and to make the annual meeting a real success, to make it worth while for guest speakers from out of the State to prepare interesting papers, and to show the proper *esprit de corps* towards our down-State confreres there should be present this year more physicians from upper New Castle County than from the rest of the State put together. And that applies to the women folks just as much as it does to the men's organization.

So, on to Dover, and show President Doctor McDaniel and President Mistress McDaniel that we appreciate their official efforts. The best way to show personal appreciation is to make a personal appearance.

THE JOURNAL congratulates two Delaware medical officials upon their recent marriages. Dr. Arthur C. Jost, executive secretary of the State Board of Health, was married on July 30th. Dr. Douglas M. Gay, director of clinics of the Delaware section of the American Society for the Control of Cancer was married on August 2d. To both of these worthies, our congratulations. And to the fortunate spouses, our felicitations.

THE JOURNAL also congratulates the Wilmington General Hospital on its recent approval, by the A. M. A., for the training of internes. This makes the second hospital in this State so recognized, the Delaware Hospital being the other.

MEDICAL SOCIETY OF DELAWARE

Officers and Committees for 1934

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Dover
First Vice-President ..... PAUL R. SMITH
Wilmington
Second Vice-President .... E. L. STAMBAUGH
Lewes
Secretary ..... W. H. SPEER
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Wilmington

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JAMES MARTIN, Magnolia

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To A. M. A.

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Alternate

C. E. WAGNER, Wilmington

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John H. Mullin, Wilmington
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SPECIAL COMMITTEES

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Committee on Medical Economics

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Committee on Criminologic Institutes

M. A. Tarumianz, Farnhurst; Stanley Worden, Dover; U. W. Hocker, Lewes.

Representative to the Delaware Academy of Medicine

W. O. LaMotte, Wilmington

IN MEMORIAM

L. HEISLER BALL, Faulkland ...Oct. 18, 1933
CECIL DEJ. HARBORDT, Dover..Nov. 6, 1933
WILLIAM H. HANCKER, Farnhurst, Dec. 29, 1933
LUTHER S. CONWELL, Camden ..Feb. 5, 1934
JOSEPH BRINGHURST, Felton ....Feb. 6, 1934
PETER W. TOMLINSON, Wilmington July 25, 1934

They have gone to rest mourned by the many to whom they have administered during times of trial. When the pain incident to all conditions of mind and body seemed beyond endurance, they came, they comforted, they relieved. Now departed, may they rest in peace.

GENERAL INFORMATION

BY-LAWS

Article III

SECTION 1. The Society shall hold an Annual Session, during which there shall be held daily General Meetings, which shall be open to all registered members and guests. This Session shall be held at such time and place as has been fixed at the preceding session by the House of Delegates.

Article X

SECTION 1. No address or paper before this Society, except those of the President, invited guests, and orators, shall occupy more than twenty minutes in its delivery; and no member shall speak longer than five minutes, nor more than once on any subject except by unanimous consent.

SECTION 2. All papers read before this Society shall be typewritten, double spaced, with wide margins. Carbon copies will not be accepted.

SECTION 3. All papers read before this Society shall become its property. Each paper shall be deposited with the Secretary immediately after reading.

SECTION 4. The deliberations of this Society shall be governed by parliamentary usage as contained in the latest edition of Roberts' Rules of Order, when not in conflict with these By-Laws.

SECTION 5. The Principles of Medical Ethics of the American Medical Association, as contained in the latest edition, shall govern the conduct of members in their relations to each other, to this Society, and to the public.

Essayists will please remember that all papers presented before the Society become the property of the Society and therefore are not to be published or submitted for publication elsewhere than in the DELAWARE STATE MEDICAL JOURNAL.

MONDAY, OCTOBER 8th, 1934

Meeting of the House of Delegates

New State House

8 P. M.

1. Call to Order.
2. Roll Call.
3. Reading of Minutes of Last Session.
4. Appointment of Committee on Nominations.
5. Reports of Officers.
   a. President.
   b. Secretary.
   c. Treasurer.
   d. Councilors.

6. Reports of Standing Committees.
   a. Scientific Work.
   b. Public Policy and Legislation.
   c. Publication.
   d. Medical Education.
   e. Hospitals.
   f. Necrology.

   Reports of Special Committees.
   a. Woman's Auxiliary.
   b. Cancer.
   c. Syphilis.
   d. Library.
   e. Criminologic Institutes.
7. Report of Delegate to the American Medical Association.
8. Unfinished Business.
9. New Business.
   a. Resolutions.
   b. Communications.
   c. Appropriations.
   d. Approval of Scientific Program.
   e. Selection of Meeting Place.
   f. Miscellaneous.
10. Adjournment.

TUESDAY, OCTOBER 9th, 1934

General Session

New State House

9:30 A. M.

Invocation:
Rev. S. B. Hiley, Pastor, Baptist Church, Dover

Address of Welcome:
Hon. Wallace Woodford, Mayor of Dover

The History and Progress of Medicine
Joseph S. McDaniel, M. D., Dover

Report of the House of Delegates

SCIENTIFIC PAPERS

1. A Preliminary Report on Tuberculosis Prevention in Delaware
   Joseph P. Wales, M. D., Wilmington
2. Gastro-intestinal Disease: Some Recognized Essential Diagnostic Procedures.
   Robert W. Tomlinson, M. D., Wilmington
3. Motion Picture: The Plastic Surgical Reconstruction of the Deformed Nose
   Keith Hospital for Plastic Surgery, New York
4. Recent Investigations on the Physiology of Gastric Secretion and Its Relation to Clinical Medicine
   Lay Martin, M. D., Baltimore

LUNCHEON

by the

KENT COUNTY MEDICAL SOCIETY

Mapledale Country Club

12:45 P. M.

GENERAL SESSION

New State House

2 P. M.

5. Treatment of Pneumonia by Means of Serum, Oxygen Tent and Pneumothorax
   Henry Jump, M. D., Philadelphia
6. Intracranial Injuries of the Newborn from the Standpoint of the General Practitioner
   P. Brooke Bland, M. D., Philadelphia
7. Medico-Legal Problems
   Hon. Daniel J. Layton, Chief Justice of Delaware
8. Pyelitis in Children
   Charles E. Wagner, M. D., Wilmington
9. Infant Feeding, with Special Reference to Lemon Feeding
   John B. Baker, M. D., Milford

OPEN HOUSE, INFORMAL

President and Mrs. McDaniel
229 State Street, South

8:30 P. M.

Members of the Society and the Auxiliary Invited

WEDNESDAY, OCTOBER 10th, 1934

GENERAL SESSION

New State House

9:30 A. M.

10. The Results of High Voltage X-ray Treatment in Metastatic Carcinoma of Bones
George C. McElfatrick, M. D., Wilmington

11. The Prevention and Cure of Cancer
Douglas M. Gay, M. D., Wilmington

12. Nasal Accessory Sinuses from the Standpoint of The General Practitioner
Emil R. Mayerberg, M. D., Wilmington

13. Faulty Body Mechanics a Factor Causing Diagnostic Errors
William Bates, M. D., Philadelphia

Election of President

LUNCHEON

by the Society

Hotel Richardson

1 P. M.

EXHIBITS

Delaware State Board of Health, Dover.

Keith Hospital, New York City.

WOMAN'S AUXILIARY

to the

MEDICAL SOCIETY OF DELAWARE

WEDNESDAY, OCTOBER 10th, 1934

Hotel Richardson

10 A. M.

Prayer
Secretary's Report ....Mrs. Gerald Beatty
Treasurer's Report,
Mrs. Charles E. Wagner
President's Report ..Mrs. Joseph McDaniel
Vice-Pres., New Castle Co. ..Mrs. Ira Burns
Vice-Pres., Kent Co. ...Mrs. W. C. Deakyne
Vice-Pres., Sussex Co.,
Mrs. E. L. Stambaugh

Committee Reports

Organization ....Mrs. George McElfatrick
Finance ..... Mrs. James W. Butler
Social .....Mrs. Raymond B. Moore
Program .....Mrs. Edgar Q. Bullock
Medical Welfare .....Mrs. Victor C. Nah
Hygeia .....
Editor .....Mrs. W. Edwin Bird
Delegate to Cleveland Convention,
Mrs. Lawrence Jones
Guest Speaker ...Mrs. W. Wayne Babcock
"What We Are Doing",
Mrs. Robert W. Tomlinson
President, Woman's Auxiliary to the American Medical Association
Recommendations
Resolutions
Adjournment

LUNCHEON

Hotel Richardson

1 P .M.

Social Afternoon

CANCER COMMENT

(*Continued from Page 211*)

vacuum is then created by partially withdrawing the piston; the needle is advanced into the tumor. The maneuver is repeated once without entirely withdrawing the needle from the mass, then the piston is released gradually. The material within the needle is forced out upon a small piece of filter paper and placed immediately in 10% formalin, or it may be smeared on a slide.

The method of frozen section finds its greatest use in guiding the surgeon at the time of operation. For example, a questionable lump in the breast may be incised and a small portion sent to the pathologist who will usually be able to return a diagnosis within five minutes. The surgeon can then continue with a conservative or radical operation as indicated. Frozen sections are also useful in guiding the surgeon during operation in cases in which conservation of tissue is important. Of course, the method necessitates the presence of a pathologist, with proper equipment somewhere near the operating room.

The modern treatment of malignant disease is predicated by much more than a diagnosis of cancer, although this is unquestionably of first importance. The selection of the form of treatment, whether by surgery, radium, or X-ray, depends quite as much upon the nature of the cancer as upon its location, extent, and the general condition of the patient. A knowledge of whether a tumor is adenocarcinoma, squamous cell carcinoma, melanoma, fibrosarcoma, etc., gives valuable aid in prognosis and makes it possible to predict the response of the tumor to the form of treatment selected.

Some years ago Broders divided squamous cell carcinoma of the lip into four arbitary grades of malignancy on the basis of microscopic appearance. Subsequently a similar grading was applied to other carcinomas. The slowest growing, most differentiated, and hence most radio resistant forms are graded as I. The most rapidly growing, least differentiated, and hence most radio-sensitive forms are graded as IV. Thus a sort of universal language has been developed by the use of which tumors may be concisely and precisely described, the treatment indicated, and the outcome predicted.

The fullest use of pathology as an aid to cancer control can best be accomplished by the complete co-operation of surgeon, radiologist, internist, and pathologist and the correlation of their data in the tumor clinic.

MISCELLANEOUS

Delaware Academy of Medicine

The facilities of the Academy Reading Room are available to all members. Mrs. Watson, the librarian, will be very glad to look up literature on any subject desired. Because of very satisfactory contact with the Surgeon-General's Library this service is unlimited. Arrangements can also be made for members living some distance from Wilmington to receive this service by mail.

The regular monthly clinical pathological conferences will begin again in October. The scientific meetings committee will announce a further program later.

In the latter part of November there will be a special meeting of the Academy with an invited lay audience, at which time an authoritative speaker on some appropriate subject relative to medical history will be heard. Further details will be published later. It is hoped that this meeting will be very well attended.

Medico-Military Symposium

The 1934 Medico-Military Symposium for Medical Department Reserve Officers of the Army and Navy will be held at the Mayo Clinic, from October 7th to 20th, both dates inclusive.

This is the Sixth Annual Inactive Duty Training Course to be held at the Mayo Clinic and will follow the plan which has proven so satisfactory in past years; that is to say, the morning hours will be devoted to attending clinics on subjects selected by the student officers, and the afternoon and evening hours given over to work in medico-military subjects. The medico-military program will be under the personal supervision of Colonel Kent Nelson, M. C., U. S. Army, Corps Area Surgeon, Seventh Corps Area,

and Captain J. B. Mears, M. C., U. S. Navy, District Medical Officer, Ninth Naval District.

The staff and faculty of the Mayo Clinic have placed their unexcelled facilities at the service of their government in the interest of preparedness and have extended an invitation to all the service to participate. The two weeks' excellent clinical post-graduate work must make a definite appeal to all who are interested in their profession, and does not incur as great a loss of time for the private practitioner that normally pertains to post-graduate work along professional lines.

The general motif of the medico-military part of the symposium will be "Public Health and Its Relation to National Defense." The problems of administration in military service present features not dealt with in private practice. In the great field of sanitation as applied to military service the medical officer has a distinct specialty. This course offers valuable and interesting training for the medical officer in all the components of our national defense. A splendid program of a thoroughly practical nature has been carefully compiled and the speakers selected for their ability to present authoritatively the subject assigned.

Application for this course of Inactive Duty Training should be made either to the Corps Area Surgeon, Seventh Corps Area, Omaha, Nebraska, or to the District Medical Officer, Ninth Naval District, Great Lakes, Illinois. Applications should state the character of the work the candidate desires to follow in the morning hours. All student officers are expected to attend and participate in the afternoon and evening sessions. Each applicant should fully understand that the invitation to accept this course of study without charge is extended by the Mayo Clinic; that the project is without expense to the government; and that one hundred hours' credit will be given those who take and complete the course. While it is desirable to attend the entire course, those whose time will not permit this may join or leave at any time and will receive credit for the hours spent in training. Uniforms are optional.

New York Academy of Medicine

The Seventh Annual Graduate Fortnight of the New York Academy of Medicine will be devoted to a consideration of Gastrointestinal Diseases. The Fortnight will be held October 22 to November 2.

Sixteen important hospitals of the city will present co-ordinated afternoon clinics and clinical demonstrations. At the evening meetings prominent clinicians from various parts of the country who are recognized authorities in their special lines of work will discuss the various aspects of the general subject.

A comprehensive exhibit of anatomical, bacteriological and pathological specimens and research material will be shown. Many of the exhibits will be demonstrated.

The profession generally is invited to attend. A complete program and registration blank may be secured by addressing Dr. Frederick P. Reynolds, New York Academy of Medicine, 2 East 103d street, New York City.

Postgraduate Assembly

The International Assembly of the Inter-State Postgraduate Medical Association of North America will be held in the Public Auditorium, Philadelphia, Pennsylvania, November 5-9, 1934. Many distinguished teachers and clinicians will appear on the program. A major list of the names of the contributors to the program, with other information, appears on page V of this Journal. All members of the Medical Society of Delaware are cordially invited to attend. Registration fee of $5.00 admits all members of the profession in good standing.

Vaccine for Infantile Paralysis

Pneumonia is next on the "public enemy" list of Dr. John A. Kolmer, the modest Philadelphia medical professor, who claims discovery of the first preventive for infantile paralysis.

He has already developed a fluid, he disclosed, which has been used effectively in immunizing rabbits from the lung inflammation, and the same vaccine has met with "a large degree of success" in experiments in humans.

While carrying on the pneumonia work, he said, he will attempt to supplement his in-

fantile paralysis vaccine with a serum to "cure" that disease after infection sets in.

After three years of experiments, Doctor Kolmer announced, they had found a fluid that could be produced cheaply, could be injected into the skin of children without fear of harmful effect and produce lifelong immunization.

Before trying the vaccine on themselves, he said, it was injected into the brains of many monkeys. The vaccinated animals showed no sign of illness when paralysis germs were injected, but their unvaccinated fellows fell victims to the virus within 5 to 7 days and became paralyzed and died.

The harmlessness of the vaccine was demonstrated, the professor explained, in repeated inoculation of the monkeys at weekly intervals for as many as ten weeks. And animals vaccinated 10 months ago are still completely immune.

Three doses of the vaccine at weekly intervals are considered sufficient by Doctor Kolmer for human immunization. Doses range from one-half to three cubic centimeters of the vaccine, which is treated with sodium ricinoleate, a chemical substance from castor beans. The virus is not killed in the fluid, but changes enough to make it safe for humans.

The spinal cord of one monkey will supply enough vaccine, he estimated, to give 50 children the required number of inoculations.

The vaccine must be used six weeks before paralysis germs attack the body in order to be effective, he said. —*A. P.*

Re: Cocomalt

Clinical tests prove conclusively that pregnancy is a drain upon the woman's calcium reserve. Very often this is manifested by softening of the bony structure, including the teeth. It is a well-known fact that pregnant women very often have decaying teeth, and even loose their teeth during pregnancy. Such women need their calcium intake increased and their ability to mobilize calcium augmented, for the drain upon calcium is so great at times as to actually abstract calcium from their bones and teeth.

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Chronic Brucellosis

Alice C. Evans, Washington, D. C. (*Journal, A. M. A.*, Sept. 1, 1934), summarizes the main facts relative to the incidence of chronic brucellosis as follows: 1. Contact with the casual organisms is of common occurrence. 2. The severity of infection is known to vary from the acute disease to a form so mild that the subject is unaware of the illness. 3. Clinical diagnosis is extremely difficult, even in severe cases. 4. Brucella infection is known to occur in animals that appear to be healthy. 5. There exists in this country a common malady—the so-called neurasthenia—which in its clinical manifestations cannot always be distinguished from chronic brucellosis. These facts challenge the right of a physician to make a diagnosis of neurasthenia—a diagnosis regarded as dishonorable by the patient, and also by his family, his employer and his friends—without considering, among other possibilities, the possibility of chronic brucellosis.

BOOK REVIEW

Manual of the Diseases of the Eye. By Charles H. May, M. D., 14th Edition. Pp. 496, with 376 illustrations. Cloth. Price, $4.00. Baltimore: William Wood & Company, 1934.

This edition, as the previous ones, is an admirable manual. It is a book for undergraduate medical students and general practitioners, and not a substitute for larger works. Common diseases are well described. The colored plates are excellent. The volume has been brought up to date with very little increase in its convenient size.

Announcement

The Radiological Society of North America will hold its next annual meeting at the Hotel Peabody, Memphis, Tennessee, December 3-7, 1934. The Medical Profession is cordially invited to attend. Further information may be obtained by addressing the Secretary-Treasurer, Dr. Donald S. Childs, 607 Medical Arts Building, Syracuse, New York.

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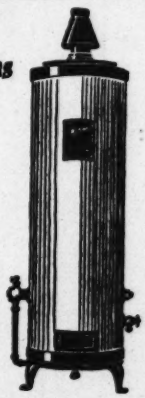

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